WHAT IS CLAIMED IS;

1. A receptor in a mammalian cell useful in the treatment of cancer which inhibits cellular activation by receptors specific for lipid-based tumor associatiated antigens.

2 The receptor of claim 1 wherein the lipid antigen is a bacterial, fungal, protozoal or mycobacterial antigen.

3. The inhibitory receptor of claims 1 and 2 wherein said inhibitory receptor contains an inhibitory receptor tyrosine-based inhibitory motifs (ITIMs).

4. The inhibitory receptor of claim 1, 2 wherein said receptor is specific for lipid-based tumor associated antigen and/or self MHC or CD1 molecules.

5. A receptor in a mammalian cell wherein said receptor inhibits cellular activation by receptors specific for lipid-based infectious disease associated antigens derived from bacteria, fungi, mycobacterium, parasite, virus, eukaryote or prokaryote antigens in the context of MHC or CD1.

6. A mammalian cell useful in the treatment of cancer wherein the inhibitory receptor for lipid-based tumor associated antigens is deleted or functionally deactivated.

7. A mammalian cell useful in the treatment of cancer wherein inhibitory receptor tyrosine-based inhibitory motifs of the inhibitory receptor for lipid-based tumor associated antigens are deleted or functionally deactivated.

8. A mammalian cell useful in the treatment of cancer wherein the the inhibitory receptor for superantigens associated with self antigens are functionally deleted.

9. A mammalian cell useful in the treatment of cancer wherein the inhibitory receptor for tumor associated lipid antigens and superantigens are deleted or functionally deactivated.

10. The lipid-based tumor associated antigens of claims 1, 2, 4, 6, 9, wherein said lipid-based tumor associated antigen is selected from the group consisting of glycolipids, proteolipids, glycosphingolipids, sphingolipids, gangliosides, phytoglycolipids.

11. The lipid antigens derived from bacteria, mycobacteria, fungi and protozoa marine invertebrates of claim 2 wherein said lipid antigens are selected from the group consisting of glycosylceramides, glycolipids, proteolipids, glycosphingolipids, gangliosides and sphingolipids with inositolphosphate-containing head groups, phytoglycolipids, mycoglycolipids, lipoarabinan and mycolic acid.

12 The sphingolipid antigens of claim 11 wherein the sphingolipid contains inositolphosphate-containing head groups with the general structure of ceramide-P-myoinositol-X with X referring to polar substituents consisting of ceramide-p-inositol-mannose, inositol-1-P-(6)mannose(a1,2inositol-1P-(1)ceramide, (inositol-P)2-ceramide, inositol-P-inositol-P-ceramide, inositol-P-inositol-P-ceramide.

13. The mammalian cell of claim 6-9 wherein said cell is an immunocyte selected from a group consisting of T cell, NK cell, NKT cells

14. A mammalian cell of claim 7 wherein the superantigen is selected from a group consisting of a staphylococcal enterotoxin, a streptococcal pyrogenic exotoxin, mycoplasma arthitides, rabies antigen, clostridial product.

15. A mammalian cell useful in the treatment of cancer wherein the inhibitory receptor for glycan-based tumor associated antigens is deleted or functionally deactivated.

16. The glycan antigens of claim 15 wherein said glycan antigen is selected from the group consisting of peptidoglycans or glycan phosphotidylinositol (GPI) structures.

17. A mammalian cell useful in the treatment of cancer wherein the the inhibitory

receptor for superantigen-associated self antigens are functionally deleted or inactivated.

18. The self antigens of claims 17 wherein said self antigens consist of a MHC or CD1 molecule.

19. A mammalian cell useful in the treatment of cancer wherein the inhibitory receptors and/or immune receptor tyrosine based inhibitory motifs which inhibits cellular activation by receptors specific for lipid-based tumor associated antigens and superantigens are deleted or functionally deactivated.

20. The superantigen of claims 17 wherein said superantigen is selected from a group consisting of the staphylococcal enterotoxins SEA, SEB, SEC, SEC1, SEC2, SEC3, SED, SEE, TSST-1 or streptococcal pyrogenic exotoxins, mycoplasma arthritides, rabies virus, mammary tumor virus, clostridial antigen.

21. A mammalian cell in which the inhibitory receptor for lipid-based infectious disease associated antigens and/or immune receptor tyrosine based inhibitory motifs which inhibits cellular activation by receptors specific for lipid-based infectious disease associated antigens derived from bacteria, fungi, mycobacteria, parasite, virus, eukaryote or prokaryote antigens are deleted or functionally deactivated.

22. The mammalian cell of claims 13, 14, 15, 17, 18, 21 wherein said cell is an immunocyte selected from a group consisting of T cells, NK cells, NKT cells

23. The lipid antigens of claims 21 wherein said lipid-based infectious disease associated antigen or fatty acid is mycolic acid or lipoarabinan,

24 A method of treating cancer in a mammal, said method comprising inactivating or deleting inhibitory receptors or immune receptor tyrosine based inhibitory motifs in immunocytes which inhibit activating receptors specific for lipid-based tumor associated lipid antigens or superantigens.

25. A method of inactivation or deletion of receptors or ITIMs in immunocytes which inhibit cell activating receptors specific for lipid-based tumor associated antigens and superantigens comprising inactivation or deletion of nucleic acids encoding ITIMs.

26. A method for producing a tumoricidal immunocyte population in vivo said method comprising allowing a tumor associated lipid antigen and superantigen to contact immunocyte activation receptors specific for tumor associated lipid antigens and superantigens in which inhibitory receptors or ITIMs which inhibit said cell activation by receptors specific for lipid-based tumor associated antigens are inactivated or deleted.

27. A method for producing a tumoricical immunocyte population *ex vivo*, said method comprising:

a) allowing a lipid-based tumor associated antigen and superantigen to contact immunocyte activation receptors specific for lipid-based tumor associated antigens and superantigens in which inhibitory receptors or ITIMs which inhibit said cell activating receptors for lipid-based tumor associated antigens are deleted or inactivated.

b) administering said tumoricidally activated immunocytes to the host.

28. A method of producing a immunocyte population effective against infectious disease in a mammal *in vivo* said method comprising:

a) allowing a lipid-based infectious disaease associated antigen and superantigen to contact immunocyte activation receptors specific for and superantigens in which inhibitory receptors or ITIMs which inhibit said cell activation receptors specific for lipid-based infectious disaease associated antigen and superantigens are inactivated or deleted.

29. A method for producing an immunocyte population effective against infectious

disease in a mammal *ex vivo*, said method comprising:

a) allowing a lipid-based infectious disease associated antigen and superantigen to contact immunocyte activation receptors specific for lipid-based infectious disease associated antigens and superantigens in which inhibitory receptors or inhibitory receptors with tyrosine-based inhibitory motifs which inhibit said cell activating receptors for lipid-based infectious disease associated antigens are deleted or inactivated.

b) administering said immunocyte population effective against infectious disease to the host.

30. The immunocytes of claims 26-29 wherein the said immunocytes comprise a group consisting of a T cell, NK cell or NKT cell

31. The immunocytes of claim 27, 29 wherein the said immunocytes are expanded in cytokines ex vivo prior to said administration

32. The method of claims 24-29 wherein said superantigen comprises a staphylococcal enterotoxin, a streptococcal pyrogenic exotoxin, mycoplasma arthritites, rabies virus, clostridial antigen, heat shock protein.

33. The staphylococcal enterotoxin of claim 32, wherein said enterotoxin is selected from the group consisting of SEA, SEB, SEC1, SEC2, SED, SEE, SEF, TSST-1, SPEA, SPEB, SPEC, Streptococcal pyogenic exotoxin.

34. The superantigen of any of the claims wherein said superantigen is expressed by a tumor cell or accessory cell which has been transfected with a nucleic acid encoding a superantigen.

35. The superantigen of claims 34 wherein said superantigen is expressed on the surface of a cell.

36. The cell of claim 35 wherein said cell is a tumor cell or an accessory cell.

37. The superantigen transfected tumor cell or accessory cell of claims 34-36 comprising transfecting said transfected cell with additional nucleic acids selected from a group comprising an adhesion molecule, an MHC molecule, a costimulatory molecule or a plurality thereof wherein said transfected cell expresses said encoded molecule(s) from said nucleic acid.

38. The transfected tumor cell or accessory cell of claims 34-37 wherein said transfected cell is transfected *in vivo.*

39. The transfected tumor cell or accessory cell of claims 34-37 wherein said transfected cell is transfected *ex vivo..*

40. A mammalian cell wherein inhibitory receptors or their ITIMs and Fas ligand receptors are deleted or functionally inactivated

41. The mammalian cell of claim 30, 31 wherein said cell is an immunocyte selected from a group consisting of T cell, NK cell, NKT cells

42. A method of treating cancer by wherein lipid-based tumor associated antigen or superantigen agonist motifs selectively contact immunocyte activating receptors and not immunocyte inhibitory receptors *in vivo* thereby producing an immunocyte population which is effective in the treatment of cancer.

43. A method of treating cancer by wherein lipid-based tumor associated antigen or superantigen agonist motifs selectively contact immunocyte activating receptors and not immunocyte inhibitory receptors *ex vivo* thereby producing an immunocyte population which is administered to the host and is effective in the treatment of cancer.

44. A method of treating infectious disease wherein lipid based infectious disease associated antigen agonist motifs selectively contact immunocyte activating receptors and not immunocyte inhibitory receptors *in vivo* thereby producing an immunocyte population effective in the treatment of infectious disease.

45. A method of treating infectious disease wherein lipid-based infectious disease associated antigens or superantigen agonist motifs selectively contact immunocyte activating receptors and not immunocyte inhibitory receptors *ex vivo* thereby producing an immunocyte population which is administered to the host and is effective in the treatment of infectious disease.

46. A method of treating infectious disease wherein lipid-based tumor associated antigens, lipid-based infectious disease associated antigens or superantigen antagonist motifs are deleted or blocked from contact with immunocyte inhibitory receptors thereby allowing agonist motifs to stimulate immunocyte activating receptors to produce an immunocyte population which is effective in the treatment of cancer or infectious disease.

47. A method of treating cancer and infectious disease according to claims wherein the immunocytes are transfected with HSV thymidine kinase gene which induces immunocyte death in vivo in response to exogenous administration of gancyclivir.

48. An mammalian antigen presenting cell wherein MHC class I molecules molecules of said cell are deleted or inactivated rendering said cell capable of presenting tumor associated lipid antigens and superantigens to immunocytes which are then capable of inducing a tumoricidal response,

49. An mammalian antigen presenting cell wherein MHC class I molecules molecules of said cell are deleted or inactivated rendering said cell capable of presenting infectious disease associated lipid antigens and superantigens to immunocytes which induce an effective response against infectious disease.

50. A mammalian cell comprising a fusion of a tumor cell with a mammalian cell whereby said fusion cell expresses glycosylceramides and tumor antigens.

51. A mammalian cell comprising a fusion of a tumor cell with a mammalian or invertebrate cell whereby said fusion cell expresses tumor antigens and phytosphingolipids.

52. The fusion cells of claims 50, 51 wherein the said fusion cells are transfected with superantigen genes whereby said fusion cell expresses a superantigen.

53. A pharmacuetical composition useful in treatment of cancer comprising a lipid-based tumor associated antigen conjugated to a superantigen.

54. The composition of claim 53 wherein the lipid-based tumor associated antigen is selected from a group consisting of a glycolipid, proteolipid, glycosphingolipid, ganglioside.

55. A pharmaceutical preparation useful in the treatment of infectious disease comprising a lipid-based infectious disease associated antigen conjugated to a superantigen

56. The composition of claim 55 wherein the infectious disease associated lipid antigen is selected from a group consisting of a glycolipid, proteolipid, glycosphingolipid, ganglioside , phytosphingolipid, mycosphingolipid, lioarabinan or mycolic acid

57. The composition of claim 56 wherein the sphingolipid contains inositolphosphate-containing head groups with the general structure of ceramide-P-myoinositol-X with X referring to polar substituents consisting of ceramide-p-inositol-mannose, inositol-1-P-(6)mannose(a1,2inositol-1P-(1)ceramide, (inositol-P)2-ceramide, inositol-P-inositol-P-

ceramide, inositol-P-inositol-P-ceramide.

58. A pharmacuetical composition useful in the treatment of cancer comprising a tumor associated glycan antigen conjugated to a superantigen.

59. The composition of claim 58 wherein the glycan is selected from a group consisting of a peptidoglycan or glycan- phosphotidyinositol (GPI) structures.

60. The compositions of claims 53-59 wherein the conjugates are bound to an MHC or CD1 receptor.